

**IN THE UNITED STATES DISTRICT COURT FOR THE
NORTHERN DISTRICT OF MISSISSIPPI
WESTERN DIVISION**

FRED BECK, ET AL.

PLAINTIFFS

v.

CIVIL ACTION NO. 3:03CV60-P-D

KOPPERS INC., ET AL.

DEFENDANTS

CONSOLIDATED WITH

HOPE ELLIS, ET AL.

PLAINTIFFS

v.

CIVIL ACTION NO. 3:04CV160PD

KOPPERS INC., ET AL.

DEFENDANTS

**DEFENDANTS' CHART OF DR. DAHLGREN'S
KEY REFERENCES FOR SHERRIE BARNES**

DR. DAHLGREN'S KEY REFERENCES FOR SHERRIE BARNES

#	Author, Title, Year	Primary Research?	Human Study?	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
1	Organochlorine compounds and estrogen-related cancers in women Adami, Hans-Olov, et al, 1995	No	Yes	Yes	Yes	Yes	No	No	Analyzed evidence linking PCB, DDE, and TCDD to breast and endometrial cancers. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.895, 10-12). The article concluded that available data does not indicate organochlorines will affect risk of these cancers except in "the most unusual situations." (Dep. V, p.896, 1.21). The article is not supportive of causation in the case of Sherrie Barnes (Dep. V, p.898, 1.19).
2	Organochlorine Compounds in Relation to Breast Cancer, Endometrial Cancer, and Endometriosis: An Assessment of the Biological and Epidemiological Evidence Ahlborg, Ulf, et al., 1995	No	Yes	Yes	Yes	Yes	No	No	Analyzed animal and human research linking organochlorines to breast and endometrial cancers. Concluded that available animal evidence does not support link between cancer and various organochlorine compounds at environmental levels, though the available data did not allow a link to be conclusively rejected. Dr. Dahlgren acknowledged that review papers are not usually relied upon to support an expert opinion because they do not contain new data (Dep. V, p.902, 11.5-13, 18, and 21).
3	Cytochrome P4501A1 and Glutathione S-Transferase (M1) Genetic Polymorphisms and Postmenopausal Breast Cancer Risk. Ambrosone, Christine, et al., 1995	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed increased breast cancer risk for women with one gene variant to those with normal allele. Found no increased risk of breast cancer except for smokers with specific gene variant.

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4	Critical Windows of Exposure for Children's Health: Cancer in Human Epidemiological Studies and Neoplasms in Experimental Animal Models. Anderson, Lucy, et al , 2000.	No	Yes	No	No	No	No	No	Analyzed the evidence that critical windows of exposure exist for human cancers. Acknowledged that the current list of environmental exposures that occur during the perinatal/postnatal period that have an association with childhood or adulthood cancers is inconsistent and inconclusive (Abstract). Stated "the cause or causes of the majority of malignancies in children and young adults remain unknown." (Abstract). Dr. Dahlgren acknowledged the article's statement that "[t]he evidence for exposures occurring during the preconceptional period that have an association with childhood or adulthood cancers is equivocal." (Dep. V, p.909, 1.23). Analyzed PAHs from soil samples at two locations for impact on human breast cancer cells. Dr. Dahlgren acknowledged that the study was comprised of studying cells in a petri dish (Dep. V, p.914, 1.16).
5	Antiestrogenicity of environmental polycyclic aromatic hydrocarbons in human breast cancer cells. Arcaro, Kathleen, et al., 1999	Yes	Yes	No	No	Yes	No	No	Analyzed the various environmental and health risks associated with the Seveso, Italy accident. Found decreases in the risk of breast cancer among people exposed to TCDD and related chemicals. Dr. Dahlgren acknowledged that the article does not find an increased incidence of breast cancer in multiple zones analyzed (Dep. V, p.988, ll.10-14).
6	Chemical, Environment, and Health Aspects of the Seveso, Italy, Accident. Bertazzi, Pier, et al., 1994.	No	Yes	No	Yes	No	No	Yes	Analyzed the various environmental and health risks associated with the Seveso, Italy accident. Found decreases in the risk of breast cancer among people exposed to TCDD and related chemicals. Dr. Dahlgren acknowledged that the article does not find an increased incidence of breast cancer in multiple zones analyzed (Dep. V, p.988, ll.10-14).

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7	Developmental Effects of Dioxins and Related Endocrine Disrupting Chemicals. Birnbaum, Linda, 1995.	No	No	No	No	No	No	No	Analyzed the developmental effect associated with TCDD and related PAH exposure in humans. Article does not relate to breast cancer. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.1050, l.24) The article does not relate to breast cancer (Dep. V, p.1051, ll.2-12). The article does not discuss any risks for breast cancer (Dep. V, p.1051, l.15).
8	Cancer and Developmental Exposure to Endocrine Disruptors. Birnbaum, Linda, et al., 2003.	No	Yes	No	No	No	No	No	Analyzed various studies on the relation between human and animal exposure to various toxicants (during rapid growth and differentiation) and an increased susceptibility to cancer. Dr. Dahlgren acknowledged that: The article is not an original research paper but rather summarizes other people's published works (Dep. IV, p.689, l.8). The article does not provide relative risk data (Dep. IV, p.693, l.12). The article is not a case-control study or a cohort study (Dep. IV, p.693, ll.15-18). Linda Birnbaum does very little of her own research and spends little time in the lab (Dep. VI, p.694, ll.4-7). The article referred to different types of endocrine disruptors (Dep. IV, p.695, ll.7-10). The article does not document any of the exposure levels which are required to produce mammary tumors (Dep. IV, p.696, l.4).

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9	Prenatal TCDD and Predisposition to Mammary Cancer in the Rat. Brown, Nadine et al., 1998.	Yes	No	No	No	No	No	No	<p>Analyzed prenatal exposure to TCDD and related chemicals and its correlation to mammary cancer.</p> <p>Found that prenatal exposure to TCDD does not significantly affect gland size or cell proliferation.</p> <p>Indicated that neither ecological data nor occupational studies provide clear support for an association between organochlorine endocrine disruptor exposure and occurrence of breast cancer. Nevertheless, Dr. Dahlgren claimed in his expert report that this study indicated a causal link between breast cancer and the chemicals of interest in this case. (Expert Report, p.116).</p> <p>Indicated that postnatal exposure may have a protective effect against mammary cancer.</p> <p>Dr. Dahlgren agreed with the article statement that:</p> <p>For every report of dioxin being associated with breast cancer, there seems to be one that finds no significant effect (Dep. IV, p.699, ll.11-12).</p> <p>It may be possible that postnatal exposure to TCDD may render a protective effect against mammary cancer (Dep. IV, p.700, ll.18-25; p.701, ll.1-5).</p> <p>Analyzed cancer risk of people possibly exposed to creosote living in and around a specific Missouri community.</p> <p>It is an unpublished article that warns that its conclusions should be viewed with extreme caution because: (1) exposure to creosote had not been documented in the study area population; and (2) any significant health effects noted in the article did not take into account critical confounding variables, i.e. exposure to low level air pollutants in an industrial area or past exposure history / geographical location.</p> <p>Table 3 supports the proposition that living near a</p>
10	Health Profile For Forest Products Division Facility Kerr-McGee Chemical Corporation Kansas City, Missouri. Burns & McDonnell Waste Consultants, 1992.	Yes	Yes	No	No	No	Yes	No	

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									<p>creosote plant does not cause breast cancer.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The study found lower rates of breast cancer than expected when it compared its data with both state and county data (Expert Report, p.35).</p> <p>The study did not indicate a causal link between creosote and breast cancer (Expert Report, p.116).</p> <p>"There is a relative paucity of research on the health effects of coal tar creosote." (Expert Report, p.29). In addition, he stated, "the chemical composition of studied creosotes is rarely reported." (Expert Report, p.30).</p> <p>The study was never published (Dep. VI, p.1090, 1.10).</p> <p>The study does not contain any data on breast cancer (Dep. VI, p.1091, 11.16-24).</p> <p>Analyzed effects of PAHs on various mouse cellular processes and on human breast cancer cells.</p> <p>Dr. Dahlgren acknowledges that:</p> <p>The study was an in vitro study of human breast cells and an in vitro study of rats and mice (Dep. V, p.923, 1.6).</p> <p>Study did not analyze the synergistic effect between TCDD and the MGP PAH mixture (Dep. V, p.63, 11.9-10).</p> <p>Study does not indicate any relative risk data for breast cancer (Dep. V, p.927, 11.18-23).</p> <p>Study is not directly related to causation of breast cancer in humans (Dep. V, p.928, 1.2).</p>
11	<p>Synergistic Activity of Polynuclear Aromatic Hydrocarbon Mixtures as Aryl Hydrocarbon (Ah) receptor agonists.</p> <p>Chaloupka, K., et al. 1993</p>	Yes	Yes	No	No	Yes	No	No	

#	Author Title Year	Primary Research?	Human Study?	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Cynosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
12	Polychlorinated Biphenyls Contamination in Women with Breast Cancer. Charlier, Corinne, et al., 2004.	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed and compared PCB levels in women suffering from breast cancer with healthy, age matched women. PCB is not an issue in the present case. Dr. Dahlgren acknowledged that the study recognized that the presence of a single identified PCB congener was the risk factor for breast cancer (Dep. IV, p.767, 1.16).
13	Health Effects on Nearby Residents of a Wood Treatment Plant Dahlgren, James, et al., 2003.	Yes	Yes	Yes	No	Yes	Yes	No	Analyzed the health affects on nearby residents who lived in the proximity of a wood treatment plant. Dr. Dahlgren acknowledged that: The study did not address increased risk of breast cancer (Dep. VI, p.1146, 1.22). All the people covered in the study were probably plaintiffs involved in a lawsuit against the wood treatment plant (Dep. VI, p.1153, 11.5-9). The study was a cross-sectional examination of the people who lived near the wood treatment plant (Dep. VI, p.1154, 11.6-10). He does not know whether the people who lived in the study area had a higher risk of cancer as a result of ongoing exposure to dirt and soil in the neighborhood (Dep. VI, p.1172, 11.5-10). The study did not review the medical records of all the people in the exposed population (Dep. VI, p.1184, 11.1-4). The Cancer Prevalence Table, Table 10, of the study is based on questionnaire responses and not medical records (Dep. VI, p.1186, 11.14-20).

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14	Carcinogenesis of Mammary Gland in Rat. Dao, Thomas, 1964	No	No	No	No	Yes	No	No	Analyzed effects of polycyclic aromatic hydrocarbons and hormones on the mammary glands of rats.
15	Medical Hypothesis: Xenoestrogens as Preventable Causes of Breast Cancer. Davis, Devra Lee, et al., 1993	No	Yes	Yes	Yes	Yes	No	No	Analyzed studies to support hypothesis that environmental estrogens interact with genetic factors to produce breast cancer. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.1038, ll.12-25, p.1039, l.1). The article does not itself identify any relative risks for breast cancer based on any particular type of exposure (Dep. V, p.1039, ll.6-7).
16	Adjusting Morbidity Ratios in Two Communities Using Risk Factors Prevalence in Cases. Dean, Andrew, et al., 1988. ("Dean Study")	No	Yes	No	Yes	Yes	Yes	No	The Dean Study is a "follow-up" article to the 1980 Dusch breast cancer study of people living in and around a community in Minnesota exposed to creosote. The article analyzed relative risks from published studies rather than those associated with local cases and controls. Does not show an excess risk for breast cancer in the exposed population. In fact, it indicated that the observed breast cancer incidence in the town near the wood treating plant is lower than would be expected. Dr. Dahlgren acknowledged that: This article, written by three of the same authors that published the 1980 Dusch Study, retracted the findings of the Dusch Study by finding no excess risk of breast cancer in the exposed community. (Dep. IV, p.783, ll.13-25; p.784, ll.1-4; Expert Report, p.32). Nevertheless, he cites both the Dusch Study and this article in Table 1 of his expert report to demonstrate that breast cancer can be associated with creosote exposure. (Expert Report, pp.31, 44).

#	Author Title Year	Primary Research?	Human Study?*	Case Control Study?	Relative Risk Data For Breast Cancer?	Statistical Significance? **	Limited to Exposures at Creosote & Penta?	Documents Level of Exposure for Cohort Study?	Comments
17	Plasma Concentrations of Polychlorinated Biphenyls and the Risk of Breast Cancer: A Congener- specific Analysis. Demers, Alain, et al., 2002.	Yes	Yes	Yes	Yes	Yes	No	No	<p>He is not aware of any published critique or criticism of the Dean paper (Dep. IV, p. 785, II.9-10).</p> <p>"There is a relative paucity of research on the health effects of coal tar creosote." (Expert Report, p.29). In addition, he stated, "the chemical composition of studied creosotes is rarely reported." (Expert Report, p.30).</p> <p>Analyzed women with breast cancer to assess the relation between breast cancer risk and concentrations of 14 PCB congeners measured in plasma lipids by high resolution gas chromatography.</p> <p>PCB is not an issue in the present case.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The study concluded, "although levels of these dioxin-like compounds may present a risk factor for the disease, additional studies are needed before concluding that, "these compounds are causally involved in the etiology of breast cancer." (Dep. IV, p.771, II.5-7).</p> <p>The study was not willing to commit to a definite conclusion that they have demonstrated a risk between PCB exposure and breast cancer (Dep. IV, p.771, I.12).</p> <p>The study was correct when it stated, "most studies that used the sum of all PCB congeners as the measure of exposure did not report an association with the risk of breast cancer." (Dep. IV, p.773, II.3-5).</p> <p>The study was correct when it stated, "dioxin-like compounds illicit a broad spectrum of antiestrogenic activities and may reduce breast cancer risk." (Dep. IV, p.774, II.10-16).</p> <p>The study does not identify a level of any particular PCB congener in the blood which would necessarily</p>

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									result as an increased risk. (Dep. IV, p.775, ll.24-25; p.776, ll.1-2).
18	Pesticides and Cancer Dich, Jan, et al., 1997.	No	Yes	No	Yes	No	No	No	Analyzed the international use of pesticides and evaluated the evidence that exposure to pesticides increases the risk of certain cancers in humans. Mentioned creosote only in relation to its association with skin cancer in humans. Dr. Dahlgren acknowledged that: It is a review article (Dep. V, p.928, ll.15-16). The article concluded that epidemiological studies provide limited support for an association between exposure to pesticides and the risk of cancer (Dep. V, p.929, ll.9-10). The article probably shouldn't have been included to support his opinion regarding Sherrie Barnes because it doesn't have any real data on creosote and it is not focused on breast cancer (Dep. V, p.929, ll.24-25, p.930, ll.1-3, 6-9).
19	Minnesota Department of Health Cancer Rates in a Community Exposed to Low Levels of Creosote Components in Municipal Water. Dusich, Kari, et al., 1980.	Yes	Yes	No	No	No	Yes	Yes	Analyzed elevated risk of cancer in particular Minnesota municipalities that had been exposed to PAH in their drinking water. Concluded that the elevated incidence of breast cancer cannot be attributed to water contamination. Dr. Dahlgren acknowledged that: The study does not isolate the level of creosote in the ground water which is necessary to cause an increased

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	("Dusich Study")								<p>risk of breast cancer (Dep. IV, p.781, II.21-22).</p> <p>The same authors later published a 1988 study entitled <i>Adjusting Morbidity Ratios in Two Communities Using Risk Factors Prevalence in Case</i>, which retracted the findings in this study by finding no excess risk of breast cancer in the exposed community. (Expert Report, p.32). See Dean, et al. (1988). Nevertheless, Dr. Dahlgren cites both the 1988 Dean Study and this study in Table 1 of his expert report to demonstrate that breast cancer can be associated with creosote exposure. (Expert Report, pp.31, 44, 116).</p> <p>"There is a relative paucity of research on the health effects of coal tar creosote." (Expert Report, p.29). In addition, he stated, "the chemical composition of studied creosotes is rarely reported." (Expert Report, p.30).</p>
20	Genotoxicity of Environmental Agents in Human Mammary Epithelial Cells. Eldridge, Sandra, et al., 1992.	Yes	Yes	No	No	No	No	No	<p>Analyzed and developed an assay for detecting genotoxic activity, as unscheduled DNA synthesis (UDS), induced by chemicals and UV radiation in early passage cultures of normal human mammary epithelial cells (HMEC) derived from five different women.</p> <p>Found no UDS activity with 2,3,7,8 TCDD. Article also acknowledged that the only environmental agent that has been proven to induce breast cancer in women is ionizing radiation.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The study was not a case control study (Dep. IV, p.787, II.18-19).</p> <p>The study does not contain any relative risk data for breast cancer (Dep. IV, p.787, I.25).</p> <p>The study does not indicate any statistically significant relationship between any particular exposure and breast cancer (Dep. IV, p.788, II.4-5).</p>

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21	Pesticides and Polychlorinated Biphenyl Residues in Human Breast Lipids and Their Relation to Breast Cancer. Falck, Frank, et al., 1992.	Yes	Yes	Yes	No	Yes	No	No	<p>"There is a relative paucity of research on the health effects of coal tar creosote." (Expert Report, p.29). In addition, he stated, "the chemical composition of studied creosotes is rarely reported." (Expert Report, p.30).</p> <p>Analyzed, measured and compared levels of chemical residues in mammary tissue from women with malignant and nonmalignant breast disease.</p> <p>Preliminary study that addressed compounds, including DDT and PCBs, not at issue in the present case.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The technique used by the article was an older technique that is not as accurate in terms of assessing the PCB body burden for the specific congeners (Dep. IV, p.791, ll.6-24).</p> <p>There was debate in the scientific literature about whether the study was correct (Dep. IV, p.792, ll.5-8).</p> <p>The study was looking at PCBs generally, not specific congeners (Dep. IV, p.792, ll.13-14).</p> <p>The study does not calculate relative risk (Dep. IV, p.794, ll.2-5).</p> <p>The study was trying to find out whether a specific class of compounds was a risk factor (Dep. IV, p.794, l.14).</p>
22	Aromatic DNA Adducts and Polymorphisms of CYP1A1, NAT2, and GSTM1 in Breast Cancer. Firozi, Pervez, et al., 2002.	Yes	Yes	Yes	No	Yes	No	No	<p>Analyzed the relationship between levels of aromatic DNA adducts in breast tissue and polymorphisms of specific drug-metabolizing genes.</p> <p>Suggested that smoking and genotype has an affect on breast cancer risk.</p> <p>Found that the only significant predictor of the level of DNA adducts in breast tissue was smoking.</p>

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									Dr. Dahlgren acknowledged that this study supports his opinion that Sherrie Barnes may have had a particular genetic polymorphism that increased her risk for breast cancer (Dep. V, p.935, ll.3-5).
23	Environmental Toxins and Breast Cancer on Long Island. I. Polycyclic Aromatic Hydrocarbon DNA Adducts. Gammon, Marilie, et al., 2002.	Yes	Yes	Yes	No	No	No	No	Analyzed whether PAH damage to DNA increases the risk of breast cancer. First large scale study. Does not establish causal relationship between PAHs and breast cancer. Dr. Dahlgren incorrectly claimed that study indicated a causal relationship between PAHs and breast cancer. (Expert Report, p.116). Dr. Dahlgren criticized study for not addressing polymorphisms or the big confounding factor of whether the subjects had exposure to PAHs from their proximity to busy highways (Dep. V, p.938, ll.1-25, p.939, ll.1-19, 22-25, p.940, ll.1, 3-20).
24	Elevated Risk for Male Breast Cancer After Occupational Exposure to Gasoline and Vehicular Combustion Products. Hansen, Johnni, et al., 2000.	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed the link between male breast cancer and exposure to gasoline vapors and combustion products containing PAHs, benzene, 1, 3-butadiene, 1, 2-dibromoethane and 1, 2-dichloroethane. Dr. Dahlgren acknowledged that: The study only hypothesized that the results its results might be applicable to women (Dep. IV, p.797, ll.19-22). The study is generally informative, but not directly related to the cause of Sherrie Barnes breast cancer (Dep. IV, p.798, l.8).

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									<p>The study does not calculate the relative risk for breast cancer in women (Dep. IV, p.798, 1.11).</p> <p>The study does not examine specific exposure levels, but rather looks at occupational exposure of gasoline and combustible products in general (Dep. IV, p.799, 11.5-7).</p> <p>The study stated, "these results suggest that some congeners have a protective effect on breast cancer risk, while others are associated with an increased risk." (Dep. IV, p.805, 11.21-25; p.806, 11.1-2).</p> <p>The study does not indicate what dose of any particular PCB congener is necessary to cause an increased risk of breast cancer (Dep. IV, p.806, 11.15-17).</p>
25	Tobacco Smoke Carcinogens and Breast Cancer. Hecht, Stephen, 2002	No	Yes	No	No	Yes	No	No	<p>Analyzed animal and human research regarding metabolic effects of tobacco smoke.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>Article is a review paper (Dep. V, p.948, 1.9).</p> <p>Article looks at animal studies as a mechanistic way of looking at breast cancer in humans (Dep. V, p.948, 11.24-25, p.949, 11.1-2).</p>
26	Risks Factors for Breast Cancer. Helmrich, S.P., et al., 1983.	Yes	Yes	Yes	Yes	No	No	No	<p>Analyzed risk factors for breast cancer, including age at birth of first child, not having children, late age at menarche, obesity, history of benign breast disease, positive family history of breast cancer, Jewish religion etc., in a hospital based case control study of 1185 women with breast cancer and 3227 controls.</p> <p>Does not examine chemical exposures as a risk for breast cancer.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The study is "ancient history" because it is over twenty years old (Dep. V, p.945, 1.23).</p>

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									Study is generally informative regarding non-environmental risk factors for breast cancer (Dep. V, p.947, I.20). Study does not look at chemical exposure (Dep. V, p.947, I.23-24). Analyzed 304 women with breast-related surgery and 186 controls to assess correlation between PCB congeners and breast cancer risk. PCB is not an issue in the present case.
27	Joint Effects of Nine Polychlorinated Biphenyl (PCB) Congeners on Breast Cancer Risk. Holford, Theodore, et al., 2000.	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed workers exposed to phenoxy herbicides, chlophenols, TCDD, and other polychlorinated dioxins and furans in a chemical factory in the Netherlands. Dahlgren acknowledged that the study is not informative with respect to breast cancer (Dep. V, p.968, I.9).
28	Second Follow-up of a Dutch Cohort Occupationally Exposed to Phenoxy Herbicides, Chlorophenols, and Contaminants. Hooiveld, Mariette, et al., 1998.	Yes	Yes	No	No	Yes	No	Yes	Analyzed serum samples for various organochlorine compounds from women who developed breast cancer and controls. Indicated that even though it included more women with breast cancer than most previous studies, it still has limited statistical power, making firm conclusions difficult. Dr. Dahlgren acknowledged that: The study is generally informative, but is not directly related to Sherrie Barnes (Dep. IV, p.807, II.22-23). The study does not find any increase in breast cancer (Dep. IV, p.810, II.3-4).
29	Organochlorine Exposure and Risk of Breast Cancer. Hoyer, Annette, et al., 1998.	Yes	Yes	Yes	Yes	No	No	No	

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30	Epigenetics of Breast Cancer: Polycyclic Aromatic Hydrocarbons as Risk Factors Jeffy, Brandon, et al., 2002.	No	Yes	No	No	No	No	No	Analyzed the contribution of environmental xenobiotics in the etiology of sporadic mammary neoplasia. In specific it tested breast and ovarian cancer cells to investigate whether tumorigenicity of PAH's may be attributable to disruption of BRCA-1 expression by reactive metabolites. Dr. Dahlgren acknowledged that: The article is a review paper (Dep. V, p.952, 11.1-2). No one knows whether Sherrie Barnes had a high level or a low level of BRCA-1 (Dep. V, p.952, 1.25, p.953, 1.1).
31	DNA Repair Capacity of Lymphoblastoid Cell Lines from Sisters Discordant for Breast Cancer. Kennedy, David, et al., 2005	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed sister pairs to determine whether the nucleotide excision repair pathway was deficient in breast cancer patients. Dr. Dahlgren acknowledged that the study did not analyze any toxic exposure (Dep. V, p.1026, 1.16).
32	Cancer Incidence and Mortality in Women Occupationally Exposed to Chlorophenoxy Herbicides, Chlorophenols, and Dioxins. Kogevinas, Manolis, 1993.	Yes	Yes	No	No	No	No	No	Analyzed mortality and cancer incidence in an international cohort of seven hundred one women exposed to chlorophenoxy herbicides, chlorophenols, and dioxins. Dr. Dahlgren acknowledged that the study found no increase in breast cancer (Dep. V, p.986, 1.17).
33	Human Health Effects of Dioxins: Cancer, Reproductive and Endocrine System Effects. Kogevinas, Manolis,	No	Yes	No	No	No	No	Yes	Analyzed specific human effects of dioxins, including cancer, reproductive and endocrine effects. Found that studies contained inconsistent cancer results with no specific cancer appearing to predominate. Dr. Dahlgren acknowledged that:

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	et al., 2001.								<p>The article is a review paper (Dep. IV, p.810, 1.17).</p> <p>The article is generally informative, but not particularly relevant to Sherrie Barnes (Dep. IV, p.811, 11.4-7).</p> <p>The article does not find a statistically significant increase in mortality due to breast cancer (Dep. IV, p.811, 1.11). Nevertheless, Dr. Dahlgren mentioned this increase in his expert report to support his opinion on the health affects of dioxins. (Expert Report, p.62).</p> <p>The article does not identify a particular dose level which is required to increase the risk of breast cancer (Dep. IV, p.814, 1.3).</p> <p>The article found that high levels of PCBs were associated with breast cancer risk in a subgroup of women who had the variant CYP1A1-EXON 7 polymorphism (Dep. IV, p.817, 1.6). He does not know whether or not Sherrie Barnes falls within the subgroup of women who has a variant CYP1A1-EXON 7 polymorphism (Dep. IV, p.817, 11.9-11).</p>
34	Polycyclic Aromatic Hydrocarbon-DNA Adducts in Humans: Relevance as Biomarkers for Exposure and Cancer Risk. Kriek, Erik, et al., 1998	No	Yes	Yes	No	Yes	No	Yes	<p>Analyzed the link between environmental exposure to PAHs and carcinogenic processes.</p> <p>Dr. Dahlgren acknowledged that:</p> <p>The article is only a review paper on methods (Dep. V, p.1027, 1.19, p.1032, 1.13).</p> <p>Quantification is a major concern with respect to the p-32 post labeling technique because adduct recoveries are variable and relatively minor changes in the procedures may introduce large differences in the reported adduct values (Dep. V, p.1030, 11.11-21).</p>

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35	Polychlorinated Biphenyls, Cytochrome P450 1A1, and Breast Cancer Risk in the Nurses' Health Study. Laden, Francine, et al., 2002.	Yes	Yes	Yes	Yes	No	No	No	Analyzed the interaction of PCBs with distinct polymorphisms among 367 breast cancer case control pairs. Indicated that neither PCBs (32, 33) nor the CYP1A1 polymorphism (29) alone was independently associated with breast cancer risk in the population. And that the majority of studies have concluded that PCB exposures are unlikely to be a major risk factor for breast cancer in the United States. PCBs are not an issue in this case.
36	Diagnosis of Breast Cancer: Clinical and Preclinical. Leis, Henry, 1979.	No	Yes	No	Yes	No	No	No	Analyzed the use of a patient's history, through examination, diagnostic aids, biopsy, and histologic examination to diagnose breast cancer. Does not discuss exposure to chemicals and their link to breast cancer. Dr. Dahlgren acknowledged that: The article does not talk about environmental risk factors or TCDD (Dep. IV, p.818, ll.18-23). The article is not very informative with respect to causation (Dep. IV, p.819, ll.5-7).
37	Aromatic DNA Adducts in Adjacent Tissues of Breast Cancer Patients: Clues to Breast Cancer Etiology. Li, Donghui, et al., 1996.	Yes	Yes	Yes	Yes	No	No	No	Analyzed whether environmental carcinogen exposure may be involved in human breast cancer etiology. Acknowledged that radiation is the only environmental factor that has been definitely implicated in breast cancer. Dr. Dahlgren acknowledged that the study does not identify the sources of the environmental exposures it references (Dep. V, p.1049, ll.3-6).

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38	DNA Adducts in Normal Tissue Adjacent to Breast Cancer: A Review. Li, Donghui, et al., 1999.	No	Yes	Yes	No	No	No	No	Analyzed the levels of DNA adducts in normal breast tissue of 87 breast cancer patients and 29 non-cancer control patients. Acknowledged that there is no clear link between environmental factors such as cigarette smoking, dietary fat intake, and exposure to pesticide and breast cancer. Dr. Dahlgren acknowledged that: The article was a review paper (Dep. V, p.1053, 1.20). The article does not contain any relative risk data for cancer (Dep. V, p.1055, 11.1-21).
39	Genetic and Environmental Determinants on Tissue Response to in Vitro Carcinogen Exposure and Risk of Breast Cancer. Li, Donghui, et al., 2002.	Yes	Yes	Yes	Yes	Yes	No	No	Analyzed whether genetic susceptibility to carcinogen exposure is a risk factor for breast cancer by conducting a <i>small</i> scale case-control study to demonstrate individual variability in tissue response to carcinogen insult. It also analyzed the genetic and environmental factors that affect such variability. Indicated that it operated under an unbalanced design because it had limited accessibility to breast tissue and was restrained to pre-menopausal women and its controls were over represented with young minorities. This unbalance raised the question of whether the higher levels of adducts detected in cases that in controls was a consequence of the difference in age and ethnic distributions between the two groups. Found that family history of breast cancer and pack-years of smoking were significantly associated with the level of BP-induced DNA adducts in breast tissues among case subjects. Does not evaluate creosote or dioxin exposure. Dr. Dahlgren acknowledged that the study was an in vitro study (Dep. V, p.954, 1.3).

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40	Characterization of a Major Aromatic DNA Adduct Detected in Human Breast Tissues. Li, Donghui, et al., 2002.	Yes	Yes	No	No	No	No	No	Analyzed and attempted to characterize a major bulky DNA adduct detected in normal adjacent tissues of breast cancer patients. This included in vitro studies on human cells from cancer patients and animal studies. Does not evaluate creosote or dioxin exposure. Dr. Dahlgren acknowledged that he does not know whether Sherrie Barnes had the particular DNA adduct in her breast tissue that is analyzed in the study (Dep. V, p.1059, 1.6).
41	Breast Cancer Risk Factors: PCB Congeners. Lucena, RA, et al., 2001.	Yes	Yes	No	Yes	No	No	Yes	Analyzed the possible relationship between PCBs and breast cancer. Indicated that article had difficulty obtaining samples from healthy women, which resulted in hospital control subjects that may not have been representative of the general population. PCB is not an issue in the present case. Dr. Dahlgren acknowledged that: The only congener or PCB that was found to significantly increase the risk of breast cancer was no.28 (Dep. IV, p.820, 1.13). The study does not identify a particular dose level for congener no. 28, which would result in increased risk.
42	Cohort Mortality Study of Capacitor manufacturing Workers, 1944-2000. Mallin, Katherine, et al., 2004.	Yes	Yes	No	No	No	No	No	Analyzed the mortality of Caucasian workers exposed to PCBs, chlorinated naphthalenes, and other chemicals at an electrical capacitor manufacturing plant. Acknowledged that the study suffered from a major limitation. Namely, there was difficulty assessing "exposure and potential confounders in a plant that had not only been closed, but was destroyed many years before the study, along with many of the plants records." (p.18)
									Dr. Dahlgren acknowledged that: